A166.E1552
JACC Maarch 9, 2010
Volume 55, issue 10A

VASCULAR DISEASE

THE ASSOCIATION OF COX-2 INHIBITORS AND NON-SELECTIVE NONSTEROIDAL ANTIINFLAMMATORY MEDICATIONS WITH D-DIMER IN THE MULTI-ETHNIC STUDY OF ATHEROSCLEROSIS (MESA)

ACC Poster Contributions
Georgia World Congress Center, Hall B5
Monday, March 15, 2010, 9:30 a.m.-10:30 a.m.

Session Title: Vascular Biology/Atherosclerosis I
Abstract Category: Vascular Biology/Atherosclerosis/Thrombosis/Endothelium
Presentation Number: 1165-346

Authors: *Stewart G. Allen, Mary Cushman, David Sane, Craig Johnson, David Herrington, Wake Forest University School of Medicine, Winston-Salem, NC*

Background: Cyclooxygeanse-2 (COX-2) inhibitors and non-selective nonsteroidal anti-inflammatory medications (NSAIDs) are associated with increased cardiovascular risk. COX-2 inhibition may lead to an imbalance in prostanoids producing a prothrombotic state, thereby increasing cardiovascular events. Limited data exist on associations of these medications with biomarkers of thrombosis.

Methods: In a cross-sectional analysis of participants in Multi-Ethnic Study of Atherosclerosis (MESA), we used multiple regression models adjusting for age, gender, race, total cholesterol, hypertension, diabetes mellitus, BMI, cigarette smoking, and arthritis to examine the association between use of celecoxib (n=235), rofecoxib (n=163), and non-selective NSAIDs (n=1121) and levels of D-dimer, fibrinogen, von Willebrand Factor (vWF), Factor VIII, and PAI-1 compared with those not using COX-2 inhibitors or non-selective NSAIDs (n=5180). In secondary analysis we compared levels in users and non-users matched on propensity scores based on age and arthritis.

Results: After adjustment for covariates, D-dimer levels were significantly higher in users of celecoxib (0.65+/-0.05), rofecoxib (0.51+/-0.06), and non-selective NSAIDs (0.40+/-0.03) compared with non-users (0.35+/-0.01); (p=0.01, p=0.02, p=0.05, respectively). Participants taking celecoxib at high doses (>250mg daily) had significantly higher D-dimer than those taking lower doses (<150mg daily) (1.45+/-0.34 vs 0.28+/-0.30, p=0.02). In stratified analyses the association of COX-2 inhibitor and non-selective NSAID use with elevated D-dimer levels was not significant in users of aspirin. The association of COX-2 inhibitor use with elevated D-dimer was also evident in the propensity score matched analyses (COX-2 users: 0.65+/-0.07 vs non-users: 0.37+/-0.07; p=0.0006). COX-2 inhibitor use was not associated with the other biomarkers investigated.

Conclusions: The current analysis supports the hypothesis that increased thrombotic potential may explain part of the cardiovascular risk associated with COX-2 inhibitors and non-selective NSAIDs.